tolic volumes (EDV & ESV) is important for assessment of severity, prognosis, and efficacy of interventions in dilated cardiomyopathy.

Methods: We examined the accuracy of 2-dimensional (2D) echo (E) in measurement of LV EDV, ESV and ejection fraction (EF) in 11 pts with dilated LV and systolic dysfunction (EF 37 ± 8%. E EDV, ESV, and EF were measured using biplane disc summation method before and after intravenous injection of Albunex (A, 0.22 ml/Kg/min) with and without color Doppler (CECD) enhancement. The findings were compared with measurements obtained by multislice short-axis gradient echo magnetic resonance imaging (MRI).

Results: 2D E EDV, ESV and EF correlated well with the MRI measurements especially with CECD imaging [table].

	EDV		ESV		EF	
	r	p	r	p	r	p
2D	0.75	0.008	0.82	0.002	0.65	0.03
A	0.80	0.003	0.87	0.0006	0.89	0.0003
CECD	0.87	0.001	0.95	0.0001	0.87	0.0009

r- and p-values versus MRI

Compared to MRI, CECD had a mean standard error of estimate of: 22.7 ml (EDV), 13.3 ml, (ESV) and 5.4% (EF).

Conclusion: Color Doppler cardiography of left ventricle following LV opacification after intravenous injection of A can significantly improve the accuracy of 2D E in measurement of LV volumes and EF in dilated hearts.

1173-36 Evidence for Marked Disproportionate Myocardial Dysfunction in the Septum in Hypertensive Left Ventricular Hypertrophy

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High relative wall thickness in hypertensive left ventricular hypertrophy (LVH) has been shown to allow preserved shortening at the endocardium despite depressed LV midwall shortening (MWS) by echocardiography (Echo). Whether this finding is related to global geometric factors or local depression of myocardial shortening is unknown. Magnetic resonance (MR) tagging permits evaluation of intramyocardial strain (ϵ) (independent of LV geometry. We evaluated 19 patients (pts) with LVH ECG criteria in the LIFE study (14 m and 5 f, 52–78 yrs). Additionally, we evaluated 8 normal volunteers (vol), with no evidence of LVH, (7 m and 1 f, 25–38 yrs). Normalized LV mass (LV/ht$^{2.7}$) in pts was 57 ± 19 vs vol 27 ± 8 ($p < 0.001$) and fractional shortening in pts was 34 ± +10% vs 40 ± 4% in vol (p = ns). Peak systolic stress was within normal range in all subjects. MR ϵ was measured in a short-axis slice at the tips of the papillary muscle. The mean MR radial ϵ in pts (14 ± 11) was depressed compared to vol (25 ± 11, $p < 0.05$) as was echo MWS (14 ± 4% vs 21 ± 3%, $p < 0.001$). However, in pts vs vol MR ϵ of the posterior wall did not differ (20 ± 17% vs 28 ± 17%) while septal MR ϵ was decreased 2.5-fold (9 ± 9% vs 22 ± 5%, $p < 0.001$). Although global MWS by echo is depressed in pts with hypertensive LVH, MRI tagging demonstrates severely depressed strain patterns localized to the septum. We postulate that depressed septal function results from differential regional wall stress and represents a novel marker for failure in hypertensive LVH.

1173-37 Is Inhaled Nitric Oxide a Better Pulmonary Vasodilator Than Sodium Nitroprusside in Patients With Advanced Heart Failure?

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Pulmonary hypertension is a frequent complication in patients with advanced heart failure and if irreversible can serve as a barrier to heart transplantation. To determine whether the selective pulmonary vasodilator nitric oxide (NO) is more effective in reducing pulmonary vascular resistance (PVR) than the nonselective vasodilator sodium nitroprusside (SNP), we compared both drugs in 18 consecutive patients with advanced heart failure. Baseline characteristics included: mean age of 52.4 ± 9.1 years, male gender in 15/18, ischemic etiology in 8/18, mean LVEF of 18 ± 5%, and mean maximal exercise oxygen consumption of 13.6 ± 2.8 ml/kg/m2. Hemodynamic measurements were recorded at baseline, after inhaling NO (80 ppm) for 10 minutes, at a second baseline, and after an infusion of SNP to a maximally tolerated dose (mean 118 ± 50 μg/kg/min).

	Baseline 1	NO	Baseline 2	SNP
Ao mean (mmHg)	85 ± 9	88 ± 13	87 ± 13	68 ± 12*
PWP (mmHg)	20 ± 8	24 ± 10*	23 ± 6	11 ± 8*
PA mean (mmHg)	33 ± 11	33 ± 11	34 ± 8	20 ± 9*
CO (Fick, l/min)	3.9 ± 1.1	3.9 ± 1.2	3.9 ± 1.1	4.7 ± 1.4*
SVR (Wood U)	21.8 ± 8	22.9 ± 10	21.9 ± 9	14.8 ± 5*
PVR (Wood U)	3.9 ± 2.5	2.5 ± 1.7*	3.2 ± 1.8	2.1 ± 1.1*

Results (mean ± SD, * = $p < 0.05$ for NO or SNP versus respective baseline) are shown in the table.

Conclusions: 1) SNP and NO caused a similar reduction in PVR (SNP = 36% vs. NO = 34% reduction, p = NS). 2) The reduction in PVR with NO was due solely to a increase in the pulmonary wedge, pressure (PWP), while the reduction in PVR with SNP occurred with an increase in CO, a decrease in PA pressure and PWP, and no change in the transpulmonary gradient. 3) NO does not appear to have greater clinical utility than SNP in evaluating the reversibility of pulmonary hypertension in patients with advanced heart failure.

1173-38 Echocardiographic Indices of Right Ventricular Dysfunction Are Strong Predictors of Events in Patients With Advanced Chronic Heart Failure

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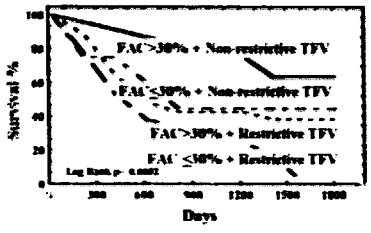
Background and Methods: In advanced chronic heart failure (HF) indices of left ventricular systolic function have a limited prognostic value. To determine whether in such patients right ventricular function provides better prognostic information, 132 sinus rhythm patients (age 54 ± 9 years) with HF due to dilated cardiomyopathy (left ventricular ejection fraction 22 ± 7%) were investigated by simultaneous right-heart side catheterization and Doppler 2-D echocardiography, performed after therapy optimization. Right ventricular end-diastolic and end-systolic areas were measured from the apical 4-chamber view and the fractional area change (FAC) was calculated. The following tricuspid flow velocity (TFV) variables were measured: peak early (Et) and late (At) diastolic velocities; their ratio (Et/At); and deceleration time (DT).



Results: During a follow-up of 27 ± 15 months cardiac events (death or urgent heart transplantation) occurred in 78 patients (59%). Both FAC and restrictive TFV were strong independent predictors (p = 0.0001 and 0.003, respectively) of events in a multivariate analysis which included clinical, hemodynamic and echo-Doppler left and right-sided variables (figure).

Conclusion: in patients with advanced HF, indices of both systolic and diastolic right ventricular dysfunction provide important prognostic information.

1173-39 Effect of ACE-Inhibition on Right Ventricular Diastolic Function in Restrictive Left Ventricular Disease

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Right ventricular function is frequently disturbed in LV disease. To assess possible effects of ACE-inhibition on right ventricular behaviour, we studied 20 pts with restrictive LV physiology, NYHA III–IV, age 62 ± 9 years, before and after symptomatic benefit by Doppler echo measurements of right ventricular systolic and diastolic free wall motion and transtricuspid flow velocities. Baseline values were compared with 21 normals with similar age.

Before ACE-I: Right ventricular systolic excursion, peak shortening and lengthening velocities were reduced compared to normals, $p < 0.001$. The onset of detectable transtricuspid flow with respect to P2 was delayed 130 ± 60 vs 30 ± 15 ms (vs normal). Peak E wave velocity was reduced and A wave velocity increased, $p < 0.001$ for each. In 7 pts mild tricuspid regurgitation demonstrated a pressure drop of 30 ± 5 mmHg in the absence of any abnormal shortening of right ventricular free wall. With *ACE-I*, mechanical right ventricular systolic and diastolic function did not change while the delayed onset of flow regressed from 130 ± 80 to 70 ± 50 ms, after P2, $p < 0.001$. Transtricuspid E wave velocity increased from 13 ± 16 to 30 ± 10 cm/s, as did E/A ratio from 0.5 ± 0.8 to 1.2 ± 0.7, $p < 00.001$ for each.

Conclusion: In the absence of free wall incoordination and only moderate elevation of right ventricular pressure, diastolic function is markedly disturbed in LV restrictive disease. Its improvement with ACE-inhibition suggests that right ventricular abnormalities result from raised left ventricular diastolic pressures.